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Comparison of cross sectional area and fat infiltration of the epaxial muscles in dogs with and without spinal cord compression

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ABSTRACT

This study investigated the cross sectional area (CSA) and fat infiltration of the epaxial muscles in Dachshunds with compressive spinal cord lesions due to intervertebral disc herniation (IVDH) and in dogs with non-compressive spinal cord lesions with fibrocartilaginous embolism (FCE). The CSA and fat infiltration of the multifidi and longissimus dorsi muscles were determined from T1 weighted magnetic resonance images. Difference in CSA and fat infiltration between the lesion- and non-lesion side in the Dachshunds was assessed using mixed model analysis. Difference in CSA and fat infiltration between Dachshunds and FCE dogs was analysed with independent sample t-tests.

There was no difference in CSA or fat infiltration between sides in the Dachshunds. FCE dogs had greater CSA (multifidus $P = 0.036$, longissimus $P < 0.001$) and less fat infiltration compared to Dachshunds (longissimus $P = 0.017$). Duration of neurological deficits, age, body size and conformation are likely to have influenced the difference between the groups.

1. Introduction

Intervertebral disc herniation (IVDH) is a common problem in companion dogs (Bergknut et al., 2012; Packer et al., 2013) with a clear breed predisposition for degeneration of the intervertebral discs (Hansen type I disc degeneration) among Dachshunds (Jensen et al., 2008). In this disease, the nucleus pulposus degenerates and mineralises. The annulus fibrosus may rupture and extrusion of the nucleus pulposus into the vertebral canal causes compression of the nerve roots or spinal cord (Hoerlein, 1952). In Dachshunds this occurs most frequently in the thoracolumbar area, accompanying signs may be acute onset of pain, paresis or plegia (Hoerlein, 1952). Research in veterinary medicine has focused on the pathogenesis (Spitzbarth et al., 2011), histopathology (Henke et al., 2013), surgical treatment (Laitinen and Puerto, 2005), recovery to ambulation (Olby et al., 2003) and prognosis for recovery (Davis and Brown, 2002; Ruddle et al., 2006).

Human low back pain (LBP) is defined as local or radiating, often non-specific pain arising from the lower part of the spine and is a frequent reason for persistent disability and early retirement (Luomajoki, 2010). Human research has used magnetic resonance imaging (MRI) (Kader et al., 2000; Kang et al., 2007) to investigate the cross sectional area (CSA) and fat infiltration of the back muscles in subjects with LBP in order to design effective management strategies for this problem (Hides et al., 1994; Mannion et al., 2009). Decrease of multifidus and erector spinae muscle CSA and fat infiltration of the muscle tissue are indicators of atrophy and associated with LBP (Kang et al., 2007; Kjaer et al., 2007). Humans with both acute (Hides et al., 1994) and chronic (Hides et al., 2008a) LBP have displayed decreased CSA in the multifidus muscle ipsilateral to the focus of painful symptoms. Asymptomatic subjects have significantly larger multifidi muscles compared to those with LBP (Hides et al., 2008a). Reduced CSA and dysfunction of the multifidus can predispose to recurrence of the symptoms (Hides et al., 2001); however, the changes may persist even though the pain has resolved (Hides et al., 1996). The multifidi are considered one of the main stabilisers of the human spine (Moseley et al., 2002, 2003) and specific training of the multifidi muscles has reduced the recurrence of LBP (Hides et al., 2001), restored the CSA of the multifidi muscles and relieved the symptoms (Danneels et al., 2000; Hides et al., 2008b).

The anatomy (Evans 1993a), innervation (Bogduk, 2005a; Kottlors and Glocker, 2008) and function (Ritter et al., 2001; Schilling and Carrier, 2009) of the canine multifidi and longissimus dorsi muscles are similar to that in humans. Multifidi muscle atrophy occurs also in horses with back pain (Stubbs et al., 2010) and one scientifically tested management strategy of back pain both in humans (Hides et al., 2008b; Mannion et al., 2009) and horses (Stubbs et al., 2011)

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Table 1
Descriptive statistics of the studied dogs.

Group	Breed (n)	Gender (n)	Lesion category (n)	Age*	Weight
IVDH	Dachshunds (52)	Male (25) Female (27)	Acute compressive (32) Chronic compressive (20)	7.3 ± 2.3	7.2 ± 2.5
FCE	Various breeds (12)[a]	Male (7) Female (5)	Acute non-compressive (12)	5.6 ± 2.4	10.1 ± 3.9

The breed, gender, lesion category, mean and standard deviation of the age and weight in the studied dogs. [a]Wales terrier (1), Border terrier (1), Bishon frisé (1), Dachshund (1), Staffordshire bullterrier (1), Whippet (1), Cocker spaniel (1), Chihuahua (1), Lhasa apso (1), German spitz (1), Mixed breed (2).
*Significant difference between IVDH and FCE groups (P = 0.021).

is specific retraining of the multifidi muscles. To date, there are no reports on atrophy, CSA or fat infiltration of epaxial muscles in dogs with or without signs of spinal cord disorders that would provide justification for therapeutic exercises.

The aim of this study was to determine the relationship between compressive and non-compressive spinal cord lesions in dogs weighing ≤15 kg on the thoracolumbar epaxial muscles. The multifidi and longissimus dorsi muscles at the level of the T10–L3 vertebrae were assessed for CSA and fat infiltration in Dachshunds with compressive spinal cord lesions, IVDH and in dogs with non-compressive spinal cord lesions diagnosed with FCE.

2. Materials and methods

2.1. Subjects

The ethics and welfare committee of the authors' institution approved this study. As there were no canine studies the sample size was approximated using information from a human study that evaluated the CSA in several spinal segments (Hides et al., 2008a, Table 1), suggesting 8–24 dogs in each group (based on 5% type I error and 80% power). Fifty-two client owned Dachshunds and 12 control dogs undergoing spinal MRI at the Royal Veterinary College (RVC) between 2003 and 2010 were retrospectively reviewed from patient records for inclusion in the study. Inclusion criteria were to be a Dachshund with a myelopathy secondary to IVDH, localised between the third thoracic and third lumbar (T3–L3) spinal cord segment and diagnosed by MRI. Dogs with other causes for T3–L3 myelopathy or evidence for previous spinal cord compression with or without surgery were excluded from the study. Control dogs were dogs of ≤15 kg with a T3–L3 myelopathy diagnosed on MRI to have a FCE and no evidence for compression of the spinal cord. All dogs had to have good quality transverse T1-weighted MRI images of the spine between the 10th thoracic and the 3rd lumbar vertebrae (T10–L3). Lesion side, lesion site, age, breed, sex, body weight, neurological grading at the time of presentation, duration of neurological deficits and duration of pain reported by the owner prior to presentation was retrieved from the patient records.

If the side of lesion was not found in the patient records, the ECVN Diplomate in the author team (ED) confirmed the side of lesion from the MRIs. Many IVDH lesions were partly ventral and it was decided to classify the lesion as 'right sided' if the lesion was right and ventral and 'left sided' if left and ventral. The lesion was classified as 'midline', only if the lesion was purely midline. The neurological grade at the time of presentation was retrieved from patient records and where not written, it was determined retrospectively based on the Modified Frankel Score used previously by Van Wie et al., 2013.

Based on the nature of the spinal cord lesion the dogs were categorised into three groups: Dachshunds with acute compressive lesions (duration of acute neurological signs less than 7 days prior to presentation), Dachshunds with chronic compressive lesions (>7 days) and other small breed dogs with acute non-compressive lesions (FCE dogs).

2.2. Methodology

All MR images were obtained with a 1.5 Tesla scanner (Phillips Intera, Phillips Medical, Reigate, UK). Segments from the T10–L3 vertebrae were analysed using a dedicated DICOM viewer (Osirix, Pixmeo, Bernex, Switzerland) from T1 sequences (TE 8–120, TR 400.00–3680.79, slice thickness 2.5–4.0 mm and gap 2.8–4.4 mm). The muscle measurements were made in random order at the level of the disc at each segment as previously reported (Kang et al., 2007). The CSA of the disc (DISC CSA) was measured in the same image as the muscular measurements (Fig. 1). The CSA of the multifidi muscles (MMCSA), the CSA of the longissimus dorsi and the iliocostal muscles (EPAXCSA) and the CSA of multifidi, longissimus and iliocostal muscles combined (MMEPAXCSA) were measured bilaterally by drawing a region of interest (ROI) around the muscle, tracing the muscle margins visible on the MRI (Fig. 1). The multifidus muscle was measured alone, whereas the longissimus dorsi muscle and the iliocostal muscle were measured together forming the epaxial muscle measurement, as it was difficult to distinguish between these muscles. To compensate for possible discrepancy in body weight and body conformation between IVDH Dachshunds and FCE dogs, a muscle to disc ratio (Kang et al., 2007) was calculated for all muscle variables (Multifidus CSA:Disc (MM:DISC), Epaxial CSA:Disc

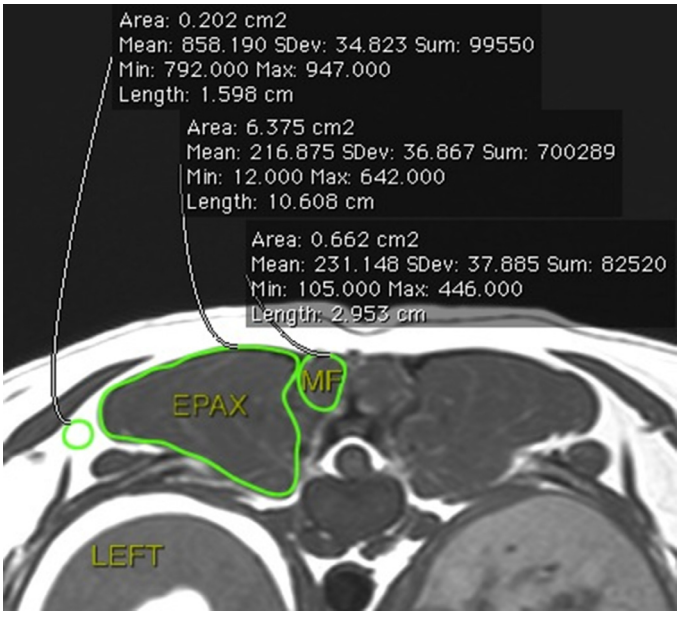

Fig. 1. The measurements. The CSA measurements on the left side at T12–13 in a FCE dog. The Muscle:Fat ratio was calculated using the hyperintensity mean of muscle and the hyperintensity mean of fat. (i.e. Muscle:Fat ratio for the left side Epaxial measurement: Epaxial hyperintensity mean (216.875)/Fat hyperintensity mean (858.190) = EPAX:FAT).

(EPAX:DISC) and Multifidus and Epaxial combined CSA:Disc (MMEPAX:DISC)).

$$\text{Muscle: Disc ratio} = \frac{\text{Muscle CSA}}{\text{Disc CSA}}$$

To evaluate the presumptive fat infiltration in the muscles, the signal intensity in the image was determined from the same ROIs. Additionally a 0.200 cm^2 large area of fat was measured at the whitest spot in the same image (Fig. 1) to ensure consistency in the intensity values because of the MRI acquisition variability. The signal intensity of muscle and fat ROI was used to calculate a muscle to fat ratio using the following formula:

$$\text{Muscle: Fat Ratio} = \frac{\text{Muscle Mean Hyper intensity}}{\text{Subcutaneous Fat Mean Hyper intensity}}$$

The Muscle:Fat ratio was calculated for all muscle measurements in all the dogs for all individual segments giving the following variables bilaterally: MM:FAT, EPAX:FAT and MMEPAX:FAT. One assessor (AFB) drew all the ROIs and was blinded to the diagnosis and background data of all the dogs at the time of the measurements.

2.3. Validation of the method

Prior to analysing the data, the measurement technique was validated. Dachshunds with IVDH (n = 6), Cavaliers with syringomyelia (n = 2), a Bishon frisé with FCE and a Pekingese with an inflammatory disease of the spinal cord were collected from patient records based on the availability to evaluate the muscles in at least four segments in the T10–L3 range. The CSA of the disc (DISCCSA), multifidus muscle (MMCSA), epaxial muscle (EPAXCSA), and combined multifidus and epaxial muscles (MMEPAXCSA) from T10 to L3 on both sides was measured in random order by the same assessor (AFB). To test repeatability, the muscles were measured twice at an interval of 3 weeks and the assessor was blinded to the earlier measurements. The reliability was good with an intra-class correlation between 0.92 and 0.99 for the muscular measurements and 0.93 for the disc CSA. The Bishon frisé and five of the Dachshunds also matched the inclusion criteria of the CSA study and were therefore later analysed together with the rest of the dogs.

2.4. Statistical analysis

Normality of the data was assessed graphically using histograms and a logarithm transformation was calculated where needed. Mean ± standard deviation was used to summarise age, body weight, durations for neurological deficits and duration for pain reported by the owner of the dogs. Statistical difference in gender, age, body weight and disc CSA between the two groups was compared with Chi Square test and independent t-tests. The difference between Muscle:Disc ratio and Muscle:Fat ratio measurements on the lesion and non-lesion side in the IVDH Dachshunds was analysed using linear mixed models to account for multiple segments from the same dog. Age, body weight, sex, breed, site of lesion, segment, neurological status at presentation, duration of neurological deficits and duration of pain reported by the owner were used as covariates to investigate their effects on the difference between sides in the muscular variables.

An independent samples t-test was performed to test the equality of means in the acute and chronic Dachshunds for the lesion and the non-lesion sides in all muscular variables, with the nature of lesion as the grouping variable and using the T12–13 segment, as most lesions occurred at this level. The same test was used to analyse differences in the disc CSA and the muscular variables between the Dachshunds and the FCE dogs at the same segment. Several of the FCE dogs had midline lesions affecting the spinal cord and categorising them into lesion and non-lesion sides was therefore inappropriate. To maintain sample size the average of the left and right side measurements was calculated in both groups for all variables; Muscle:Disc ratio and Muscle:Fat ratio using the following equation:

$$\text{Muscle: Disc ratio average} = \frac{\text{Multifidus: Disc ratio Left} + \text{Multifidus: Disc ratio Right}}{2}$$

The differences between acute compressive, chronic compressive and acute non-compressive lesions were analysed using one-way analysis of variance, (ANOVA) and Fisher's LSD post-hoc comparisons in the lesion and non-lesion side, as well as in the average variables at the T12–13 segment. Statistical significance was set at $P < 0.05$ and SPSS (version 19, IBM, New York, USA) was used for the analysis.

3. Results

3.1. Descriptive statistics

There were 52 IVDH Dachshunds and 12 FCE dogs of various breeds in this study (Table 1). The descriptive data and significant differences between groups are displayed in (Table 1). There was no significant difference in gender ($P = 0.522$) and only a trend to greater body weight in the FCE dogs ($P = 0.060$), but the age was significantly higher in Dachshunds ($P = 0.016$) (Table 1). Of the segments measured (n = 219) 10% were from T10 to T11, 19% from T11 to T12, 26% from T12 to T13, 23% from T13 to L1, 15% from L1 to L2 and 6% from L2 to L3. Mean duration of neurological deficits was 8.4 ± 16.0 days in the Dachshunds and 2.4 ± 3.9 days in FCE dogs. Mean duration of pain reported by the owner was 28.7 ± 99.1 days in the Dachshunds and 1.4 ± 4.0 days in the FCE dogs.

3.2. Difference between sides in the Dachshunds

In the Dachshunds (n = 52) the mixed model analysis showed no difference between the lesion and non-lesion side in the EPAX:DISC ratio ($P = 0.656$) and the MMEPAX:DISC ratio ($P = 0.790$). 'Duration of neurological deficits' had a significant effect on the EPAX:DISC variable ($P = 0.029$). There was no significant effect of 'site of lesion' ($P = 0.149$) or 'segment' ($P = 0.661$) on the difference between sides in the EPAX:DISC variable or in the MMEPAX:DISC variable ($P = 0.079$) and ($P = 0.698$).

The difference between lesion and non-lesion sides for the MM:FAT ratio ($P = 0.510$), EPAX:FAT ratio ($P = 0.298$) and the MMEPAX:FAT ratio ($P = 0.960$) in the Dachshunds were not significant. The 'site of lesion' had no significant effect on the difference between sides in the EPAX:FAT ($P = 0.944$) or MMEPAX:FAT variables ($P = 0.492$) nor had 'segment' significant effect on the EPAX:FAT ($P = 0.935$) or MMEPAX:FAT variables ($P = 0.876$). The covariate 'neurological grade at presentation' had a significant effect on the difference between sides in the MM:FAT ratio variable ($P = 0.040$). 'Duration of pain reported by the owner' had a significant effect on the difference between sides in the EPAX:FAT ratio variable ($P < 0.001$). The t-test analysis showed no difference between the lesion side and non-lesion side in acute or chronic Dachshunds for any of the Muscle:Disc or Muscle:Fat variables.

3.3. Difference between Dachshunds and FCE dogs

There was no significant difference in the disc CSA between Dachshunds (1.16 ± 0.28 cm^2) and FCE dogs (1.06 ± 0.36 cm^2) ($P = 0.150$). The average Muscle:Disc ratio were significantly lower

Table 2
The difference between Dachshunds and FCE dogs.

Average variables	Group	All dogs[a]		Dogs with lesion at T12–13 segment[A]	
		Mean ± sd	P	Mean ± sd	P
Muscle:Disc ratio average (cm^2)					
Multifidus	IVDH	0.25 ± 0.08	0.036	0.25 ± 0.07	<0.001
	FCE	0.59 ± 0.50		0.45 ± 0.95	
Epaxial					
	IVDH	2.28 ± 0.84	<0.001	2.48 ± 0.91	<0.001
	FCE	4.22 ± 1.24		4.06 ± 0.59	
Multifidus + Epaxial					
	IVDH	3.55 ± 1.31	<0.001	3.89 ± 1.43	<0.001
	FCE	6.87 ± 1.92		6.61 ± 0.97	
Muscle:fat ratio average mean hyperintensity					
Multifidus	IVDH	0.56 ± 0.18	0.072	0.56 ± 0.12	0.022
	FCE	0.49 ± 0.11		0.47 ± 0.06	
Epaxial					
	IVDH	0.55 ± 0.22	0.017	0.54 ± 0.15	0.038
	FCE	0.46 ± 0.07		0.44 ± 0.07	
Multifidus + Epaxial					
	IVDH	0.56 ± 0.06	0.015	0.55 ± 0.14	0.021
	FCE	0.47 ± 0.06		0.45 ± 0.06	

[a]n = 45 for IVDH Dachshunds and n = 12 for FCE dogs; [A]n = 18 for IVD Dachshunds and n = 8 for FCE dogs. Mean, standard deviation (sd) and statistical differences (independent sample t-test) between the two groups for the average variables at the T12–13 segment.

in the Dachshunds compared to FCE dogs, MM:DISC P = 0.036, EPAX:DISC P = < 0.001, MMEPAX:DISC P = <0.001 (Table 2). Also the average Muscle:Fat ratio in Dachshunds and FCE dogs revealed a significant difference in EPAX:FAT ratio (P = 0.017) and MMEPAX:FAT ratio (P = 0.015) with the intensity means being higher in the Dachshunds indicating more fat in their muscles (Table 2). The results were similar when the lesion segment was restricted to T12–13, with a significant higher MM:FAT ratio in Dachshunds than FCE dogs (P = 0.022) (Table 2).

3.4. Difference between compressive and non-compressive lesions

In the EPAX:DISC and MMEPAX:DISC variables there was a significant difference between the Dachshund group and the FCE group, on both the lesion side (P < 0.001), and the non-lesion side (P < 0.001). The post hoc analysis revealed significant differences between both the acute (P < 0.001) and the chronic (P < 0.001) compressive lesions against the acute non-compressive lesions on both the lesion and non-lesion sides for the above-mentioned variables. Similarly there was a significant difference between both the acute (P < 0.001) and the chronic (P < 0.001) compressive lesions against the acute non-compressive lesions in all averaged Muscle:Disc ratio variables (P < 0.001) and in the averaged EPAX:FAT ratio variable (P = 0.030) so that the Muscle:Disc ratio was greater and the fat infiltration was lower in dogs with non-compressive lesions. There was no difference between groups in the Muscle:Fat variables: MM:FAT lesion side (P = 0.126), non-lesion side (P = 0.141), EPAX:FAT lesion side (P = 0.206), non-lesion side (P = 0.163) and MMEPAX:FAT lesion side (P = 0.372) and non-lesion side (P = 0.147).

4. Discussion

This study found no difference in the CSA or fat infiltration between lesion and non-lesion sides in the IVDH Dachshunds. This is in agreement with a recent human study (Battié et al., 2012) on the influence of disc lesions on the multifidus CSA. However, other studies have detected asymmetry in the multifidi in humans with LBP (Hides et al., 2008a), in patients with unilateral lumbosacral radiculopathy (Hyun et al., 2007), in lumbar degenerative kyphosis (Kang et al., 2007), in pigs with experimental disc or nerve root injury (Hodges et al., 2006) and in horses with osseous spinal pathology (Stubbs et al., 2010). In dogs the spinal cord extends into the lumbosacral region (Evans, 1993b), whilst in humans, the spinal cord ends in the caudal thoracic spine (Moore and Dalley, 1999). As most protrusions in humans affect the lumbar lower vertebral discs (Bogduk, 2005b), disc herniation is more likely to compress individual nerve roots. Research has also identified signs of paraspinal denervation in humans (Yoshihara et al., 2001; Hides et al., 2008a; Hyun et al., 2007), pigs (Hodges et al., 2006) and horses (Stubbs et al., 2010) with disc herniation and nerve root compression. In dogs, IVDH usually affects the spinal cord itself (Hoerlein, 1952) and is often distributed bilaterally (Schulz et al., 1998), resulting in bilateral deficits (Besalti et al., 2005; Olby et al., 2003) and denervation atrophy. This different IVDH mechanism seen in dogs explains why unilateral multifidus CSA decrease is seen in other species, but not in these Dachshunds.

This study found no difference between sides in CSA or fat infiltration of the epaxial muscles when the acute and the chronic Dachshunds were compared. The majority of these Dachshunds were presented as emergencies with severe neurological deficits requiring immediate diagnostic workup and surgery. As such the duration of clinical signs may not have been long enough to cause significant changes in muscular CSA. Recent human research on the response of multifidus to disc herniation (Battié et al., 2012), with symptoms lasting for less than 6 weeks, reported changes in muscle composition at the lesion side and at the lesion level, but no decrease in the CSA (Battié et al., 2012). This was explained by the lack of chronicity. However, an experimental study on disc- and nerve lesions affecting the multifidus CSA in pigs reported a decrease in the CSA on the lesion side within 3 days (Hodges et al., 2006). The present study categorised acute and chronic dogs based on the duration of neurological signs, less than 7 days as acute and equal to or greater than 7 days as chronic. The analysis found the 'duration of neurological deficits' variable to have a significant effect on the EPAX:DISC ratio and the mean duration of neurological signs for chronic Dachshunds was 18.2 ± 22.3 days, which is considerably shorter than the 6–12 week long duration of LBP symptoms reported in humans (Mannion et al., 2009). Therefore it is likely that even the chronic Dachshunds were not chronic enough, or the sample size not sufficient to show asymmetry in the muscle CSA.

This study identified significantly smaller CSA in IVDH Dachshunds compared to ≤15 kg dogs with FCE. Studies in humans have identified decreased CSA in paraspinal muscles of patients with LPB compared to asymptomatic subjects (Hides et al., 2008a). The FCE dogs used for comparison in this study were not asymptomatic, but fibrocartilaginous embolism causes spinal cord infarction with acute onset of clinical signs including paresis or plegia similar to those of IVDH (Gandini et al., 2003, Nakamoto et al., 2008). However, in contrast to IVDH, the character of FCE is non-compressive and generally non-painful (Gandini et al., 2003). It is possible that the smaller CSA in Dachshunds here is a sign of denervation atrophy because of the painful and compressive character of the IVDH (Hoerlein, 1952). Previous studies have discussed denervation and disuse as possible reasons for multifidus atrophy (Hodges et al., 2006; Mattila et al., 1986). In humans with LBP, local multifidus atrophy is thought to be neurogenic (Beneck and Kulig, 2012), as disuse seems to affect the vertebral muscle CSA more generally (Belavy et al., 2011). Generalised disuse atrophy, as an explanation for the smaller CSA in the studied Dachshunds cannot be excluded. All the Dachshunds had pain and paresis of varying grades at presentation and were unable to exercise normally. The duration of neurological deficits and pain reported by the owners were noticeable longer in the Dachshunds than in the FCE dogs.

The FCE dogs were of different breeds and conformations to the Dachshunds and there was a trend to significantly smaller body weight in the Dachshunds (P = 0.060). It is also known that

vertebral morphology (Breit, 2002; Dabanoglu et al., 2004; McClain et al., 2002) and epaxial muscle architecture (Webster et al., 2014) and (Kader et al., 2000) differs between dog breeds. To standardise for any variety in muscle size between the dogs, a Muscle:Disc ratio extrapolated from human literature (Kang et al., 2007) was used. As no significant difference (P = 0.150) in the disc CSA between the Dachshunds and FCE dogs was found at the T12–13 segment the Muscle:Disc ratio was considered justified. Still, it cannot be excluded that the discrepancy in body conformation between the Dachshunds and controls has influenced the results.

An interesting finding was that the Dachshunds seem to have greater fat infiltration compared to the FCE dogs. In humans with IVDH, increased fat infiltration in the multifidus muscle ipsilateral and distal to the lesion has been identified (Battié et al., 2012), whilst those patients with LBP were found to have increased fat infiltration of the multifidus muscle bilaterally and at multiple levels (Kader et al., 2000). Duration of neurological deficits and pain reported by the owner may be associated with the fat infiltration, as progressive fat infiltration has been seen in human back muscles following denervation (Kamath et al., 2008). It is proposed that the increased fat infiltration in human spinal muscles is due to disuse (Elliott et al., 2008) and denervation (Hyun et al., 2007). Research has also shown that fat infiltration in human paraspinal muscles increases with age (Fortin et al., 2014) and is only evident in 14% of adolescents examined (Kjaer et al., 2007). In this population the age of the Dachshunds was significantly greater (7.3 years ± 2.3) than those dogs affected by FCE (5.6 years ± 2.4, P = 0.016). This may result in reduced activity levels of the Dachshunds. As such it is possible that the fat infiltration identified is age related, rather than associated with the IVDH. In humans it was also found that severe fat infiltration of the multifidus muscle was strongly associated with LBP only in adults and not in adolescents (Kjaer et al., 2007).

In agreement with recent research on human back muscles (Fortin and Battié, 2012), this study found that the CSA of canine epaxial muscles could be reliably measured using the technique employed in the presented study. Fat deposition in muscles shows high signal intensity on MRI (Cagnie et al., 2009) and T1w MRI sequences have been used successfully to evaluate fat infiltration in the human neck (Cagnie et al., 2009; Elliott et al., 2008, 2009) and back muscles (Kader et al., 2000). In this study the fat infiltration was quantified using signal intensity, as visual methods including subjective grading of the fat infiltration, have been criticised (Kang et al., 2007; Kjaer et al., 2007). A study on isolated canine infraspinatus muscles found that increased fat infiltration and decreased muscle volume on T1 weighted MR images correlated well with subsequent histological analysis (Safran et al., 2005).

The undisputable limitation of the present study is lack of a control group consisting of healthy Dachshunds, due to the ethical and financial challenges associated with this. The group used as a comparison was a heterogeneous group of dogs with FCE and was the only control population available in this retrospective study. The retrospective nature of this study brought on further limitations as the researchers had to rely on the information in the patient records only regarding the duration of pain, neurological deficits and neurological status of the dogs. Another weakness in this study is that the assessor drawing the ROIs was not blinded to the side of the disc lesion on the MR images. However, the observer did not know the history, signalment or diagnosis of the dogs at the time of the measurements.

5. Conclusions

This study found that Dachshunds with IVDH had significantly smaller CSA and greater fat infiltration compared to dogs with FCE. The results suggest future studies to consider increasing CSA and restoring muscle composition in the epaxial muscles with specific training. However, the differences in the age and body conformation between the groups must be kept in mind when interpreting the results of this work.

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